

Supportive module 2: Basics of diagnosis, treatment
and prevention of major gastroenterological diseases

Peptic Ulcer and Other Gastric and Duodenal Ulcers

LECTURE IN INTERNAL MEDICINE FOR IV COURSE STUDENTS

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US MLE TEST

A 41-year-old male who takes NSAIDs regularly for his chronic back pain develops severe abdominal pain worse with eating. Upper endoscopy is performed and the medical student asks the supervising physician how the histological differentiation between a gastric ulcer and erosion is made. Which of the following layers of the gastric mucosa **MUST** be breached for a lesion to be considered an ulcer?

1. Epithelium,
2. Epithelium, lamina propria,
3. Epithelium, lamina propria, muscularis mucosa,
4. Epithelium, lamina propria, muscularis mucosa, and submucosa,
5. Epithelium, lamina propria, muscularis mucosa, submucosa, and adventitia.

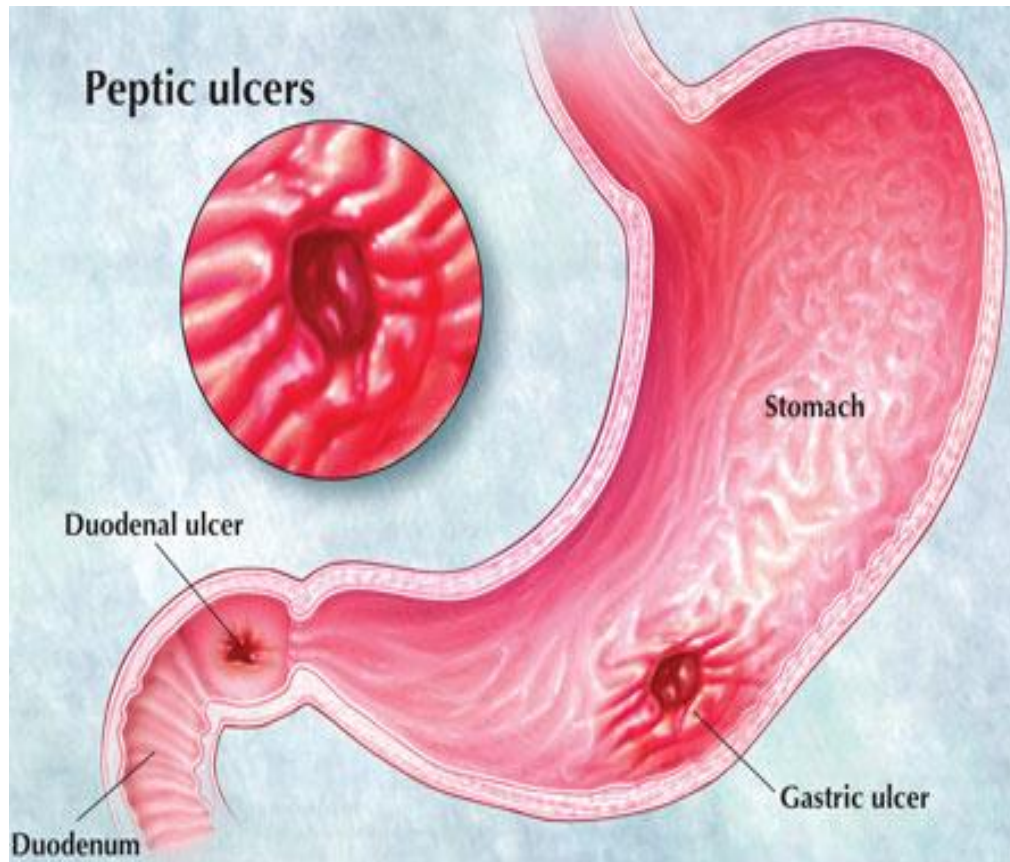
US MLE TEST EXPLANATION

The correct answer is 4. Gastric ULCERS, by definition, are a breach in the mucosa with extension into the submucosa or deeper layers. That is, the submucosa must be involved if a lesion is to be called an ulcer. In contrast, EROSIONS are mucosal defects that do NOT penetrate the muscularis mucosa.

Incorrect Answers:

1, 2: These describe an erosion which does not penetrate into the submucosa., 3: To be classified as an ulcer the submucosa must also be penetrated., 5: While deeper tissue can be breached in ulceration, the serosa or adventitia is not required to be disrupted to differentiate an erosion from an ulceration.

Plan of the Lecture



- Definition
- Epidemiology
- Mechanisms
- Classification
- Clinical presentation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic guidelines

Definition

Peptic ulcer disease (PUD) is a nonmalignant, inflammatory, mucosal lesion of the stomach or proximal duodenum in which *Helicobacter pylori* (*H. pylori*), long term use of nonsteroidal anti-inflammatory drugs and/or hypersecretion of hydrochloric acid (parietal cells) and pepsin (mucous cells and chief cells) play major etiopathogenic roles, with symptoms include epigastric discomfort (specifically, pain relieved by food intake or antacids and pain that causes awakening at night or that occurs between meals), loss of appetite, and weight loss with, with serious and life threatening complications in forms of malignancy, bleeding, perforation, peritonitis and gastric outlet obstruction.

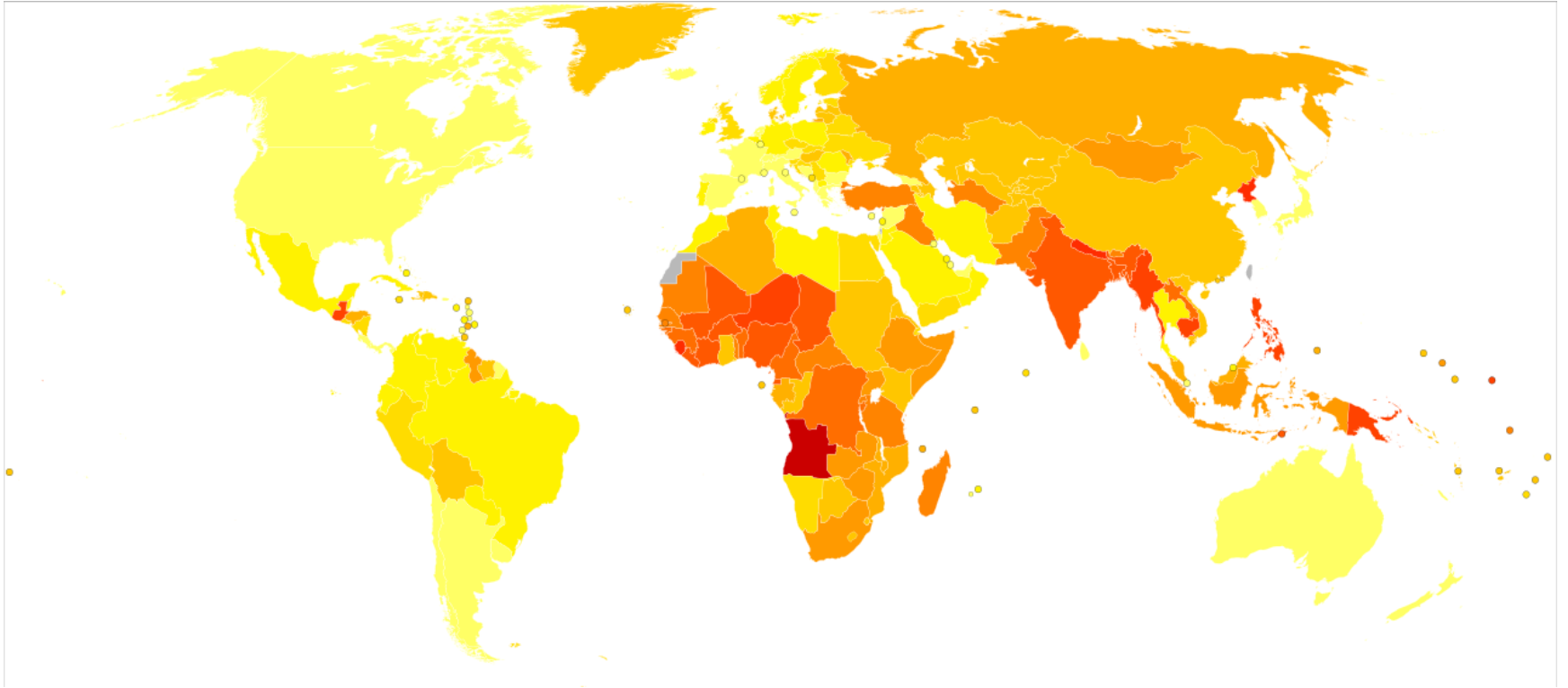
Epidemiology 1

- The lifetime risk for developing a PUD is approximately 10%
- Approximately 500,000 persons develop peptic ulcer disease in the United States each year
- In 70 percent of patients it occurs between the ages of 25 and 64 years

Epidemiology 2

- The incidence of duodenal ulcers has dropped significantly during the last 30 years, possibly as a result of the increasing use of proton pump inhibitors and decreasing rates of *H. pylori* infection, while the incidence of gastric ulcers has shown a small increase, mainly caused by the widespread use of non-steroidal anti-inflammatory drugs (NSAIDs)
- The annual direct and indirect health care costs of the disease are estimated at about \$10 billion.

Epidemiology



Disability-adjusted life year for PUD per 100,000
inhabitants

Epidemiology

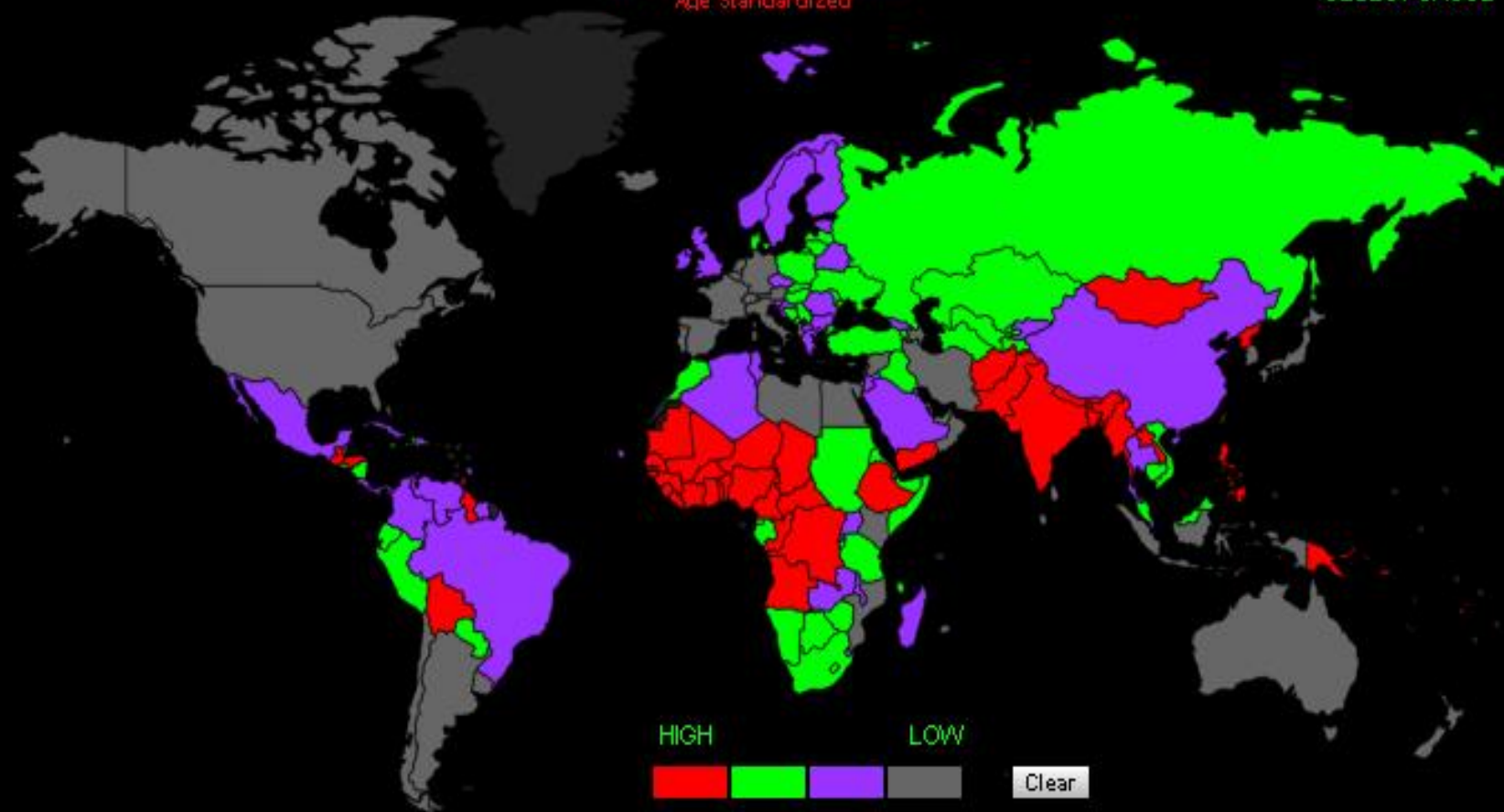
[RETURN](#) [WORLD HEALTH MENU](#)

PEPTIC ULCER DISEASE

Death Rate Per 100,000
Age Standardized

Peptic Ulcer Disease ▼

SELECT CAUSE



Risk Factors

Medical Conditions	Odds Ratio; 95% CI
Chronic obstructive pulmonary disease	(2.34; 2.21–2.47)
Chronic renal insufficiency	(2.29; 2.05–2.55)
Coronary heart disease	(1.46; 1.36–1.57)
Diabetes	(1.13; 1.06–1.20)
Behaviors	
Current tobacco use	(1.99; 1.90–2.08)
Former tobacco use	(1.55; 1.47–1.62)
Former alcohol use	(1.29; 1.22–1.37)
Demographic Characteristics	
Age >50	(1.67; 1.61–1.74)
Black race	(1.20; 1.08–1.33)
Obesity	(1.18; 1.13–1.24)
Female gender	(1.08; 1.04–1.12)
Latino ethnicity	(0.81; 0.72–0.91)

Etiology

Common Causes 1

- A major causative factor (60% of gastric and up to 50–75% of duodenal ulcers) is chronic inflammation due to *H. pylori*
- Another major cause is the use of NSAIDs: the gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by prostaglandins, but NSAIDs block the function of cyclooxygenase 1 (*cox-1*), which is essential for the production of the prostaglandins

Etiology

Common Causes 2

- Genetic factors: the lifetime prevalence of developing PUD in first-degree relatives of ulcer patients is about three times greater than the general population.

Etiology

Rare Causes 1

- Distress is well described as a cause of PUD (stress ulcers): after acute illness, multiorgan failure, ventilator support, extensive burns (Curling's ulcer), or head injury (Cushing's ulcer)
- Other medications: steroids, bisphosphonates, potassium chloride, chemotherapeutic agents (e.g., intravenous fluorouracil)

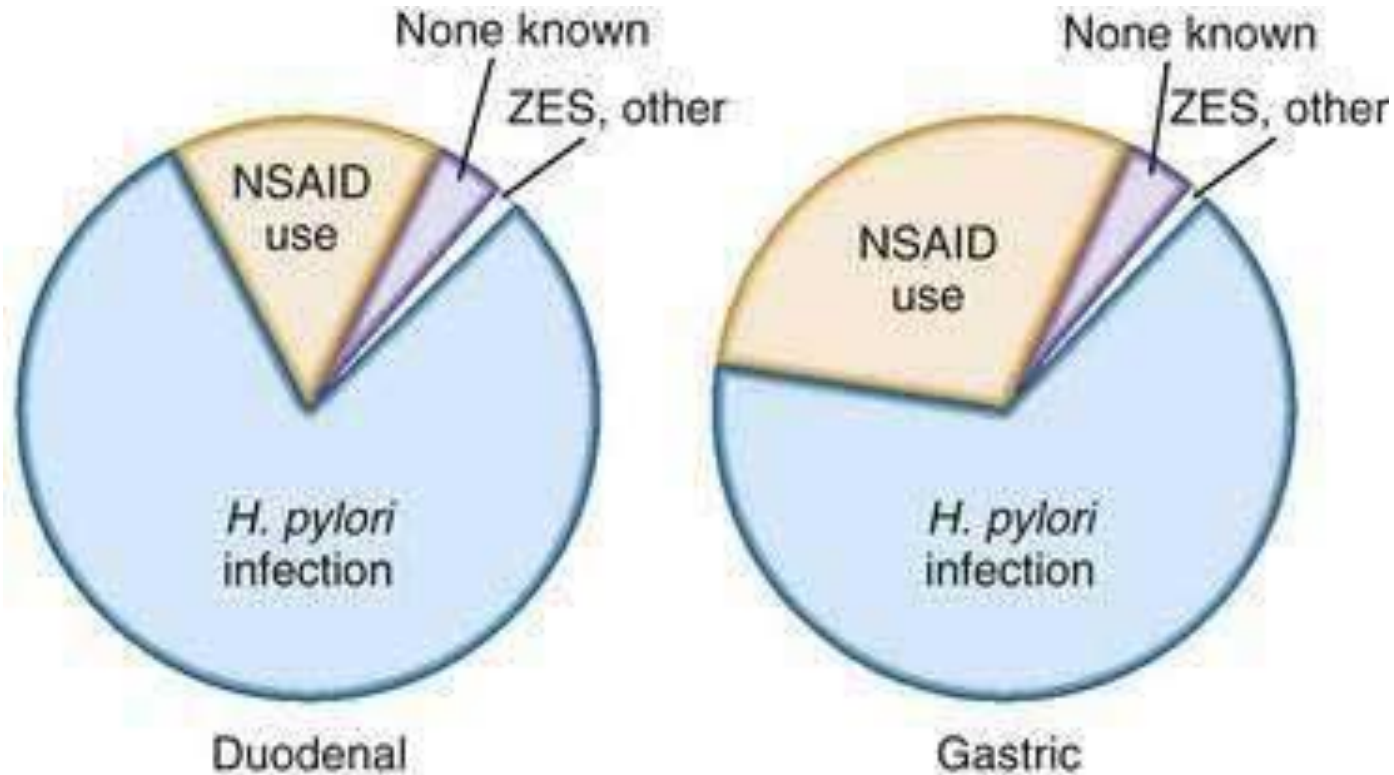
Etiology

Rare Causes 2

- Acid-hypersecretory states (e.g., Zollinger- Ellison syndrome), also cause multiple and difficult-to-heal ulcers
- Malignancy: gastric cancer, lymphomas, lung cancers.

Etiology

Associated Conditions



Pie charts depicting conditions associated with PUD in Western countries. NSAID, non-steroidal anti-inflammatory drug; ZES, Zollinger-Ellison syndrome.

US MLE TEST

A 52-year-old Caucasian male presents to your office complaining of black, tarry stool. Which of the following possible causes of this patient's presentation is LEAST associated with the development of carcinoma?

1. Barrett's esophagus,
2. H. pylori infection,
3. Adenomatous polyp,
4. Gastric ulcer,
5. Duodenal ulcer.

US MLE TEST EXPLANATION

The correct answer is 5. In contrast to gastric ulcers, duodenal ulcers are not associated with the development of carcinoma.

Incorrect Answers:

1: Barrett's esophagus is metaplasia of the esophagus that frequently progresses to adenocarcinoma., 2: H. pylori infection is a common cause of gastric ulcers and raises a patient's risk of gastric carcinoma., 3: Adenomatous colon polyps are precursors of colorectal cancer., 4: Gastric ulcers are commonly caused by H. pylori, which raises a patient's risk of gastric carcinoma.

Mechanisms

H. pylori 1

- Although *H. pylori* is present in the gastroduodenal mucosa in most patients with duodenal ulcers, only a minority (10 to 15 percent) of patients with *H. pylori* infection develop 3FB
- *H. pylori* bacteria adhere to the gastric mucosa; the presence of an outer inflammatory protein and a functional cytotoxin-associated gene island in the bacterial chromosome increases virulence and probably ulcerogenic potential

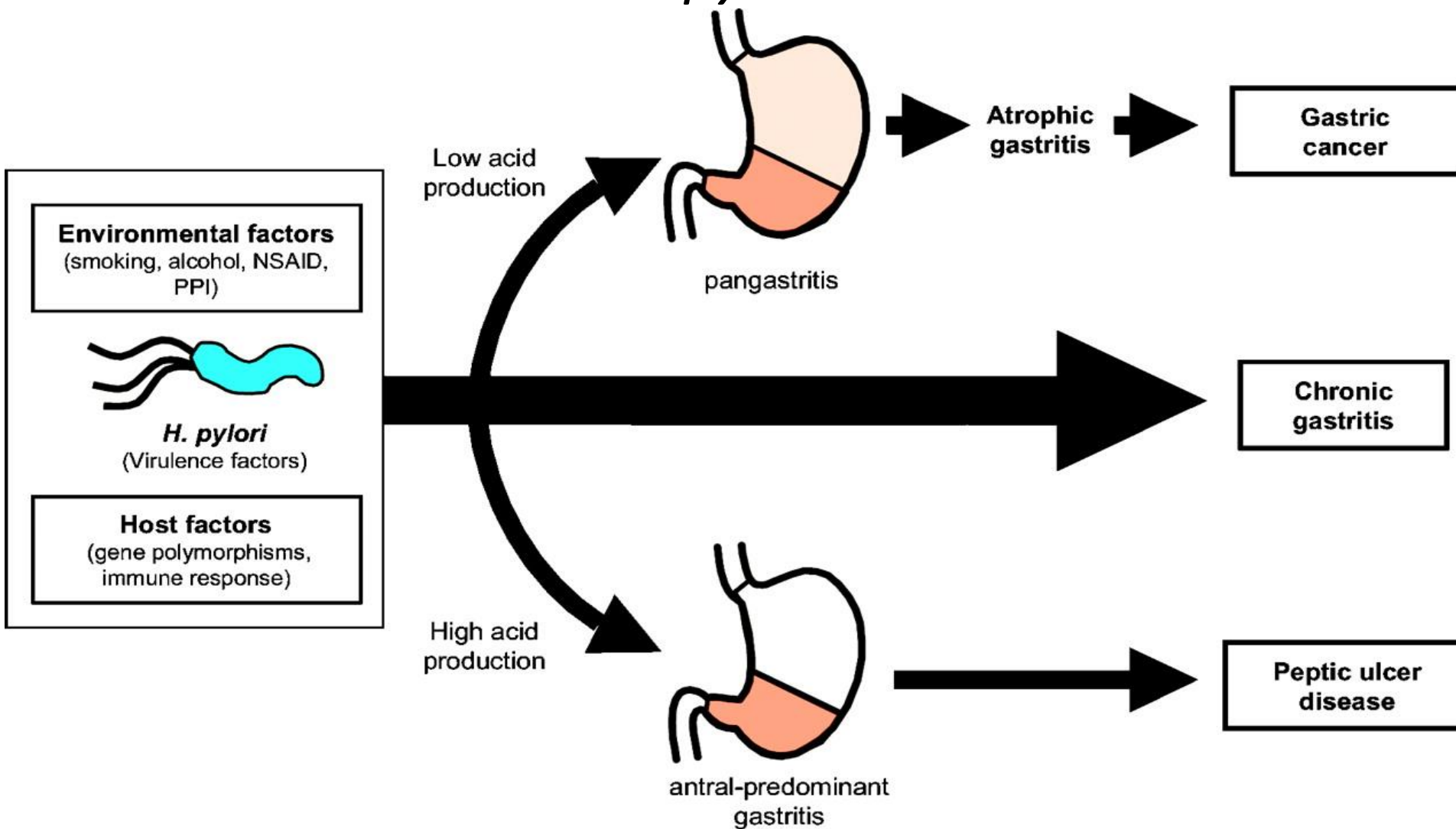
Mechanisms

H. pylori 2

- Patients with *H. pylori* infection have increased resting and meal-stimulated gastrin levels and decreased gastric mucus production and duodenal mucosal bicarbonate secretion, all of which favor ulcer formation
- Eradication of *H. pylori* greatly reduces the incidence of ulcer recurrence from 60% to 5%.

Mechanisms

H. pylori



Mechanisms

NSAIDs 1

- Topical effects of NSAIDs cause submucosal erosions
- By inhibiting cyclooxygenase, they inhibit the formation of prostaglandins and their protective cyclooxygenase-2–mediated effects (i.e., enhancing gastric mucosal protection by stimulating mucus and bicarbonate secretion and epithelial cell proliferation and increasing mucosal blood flow)

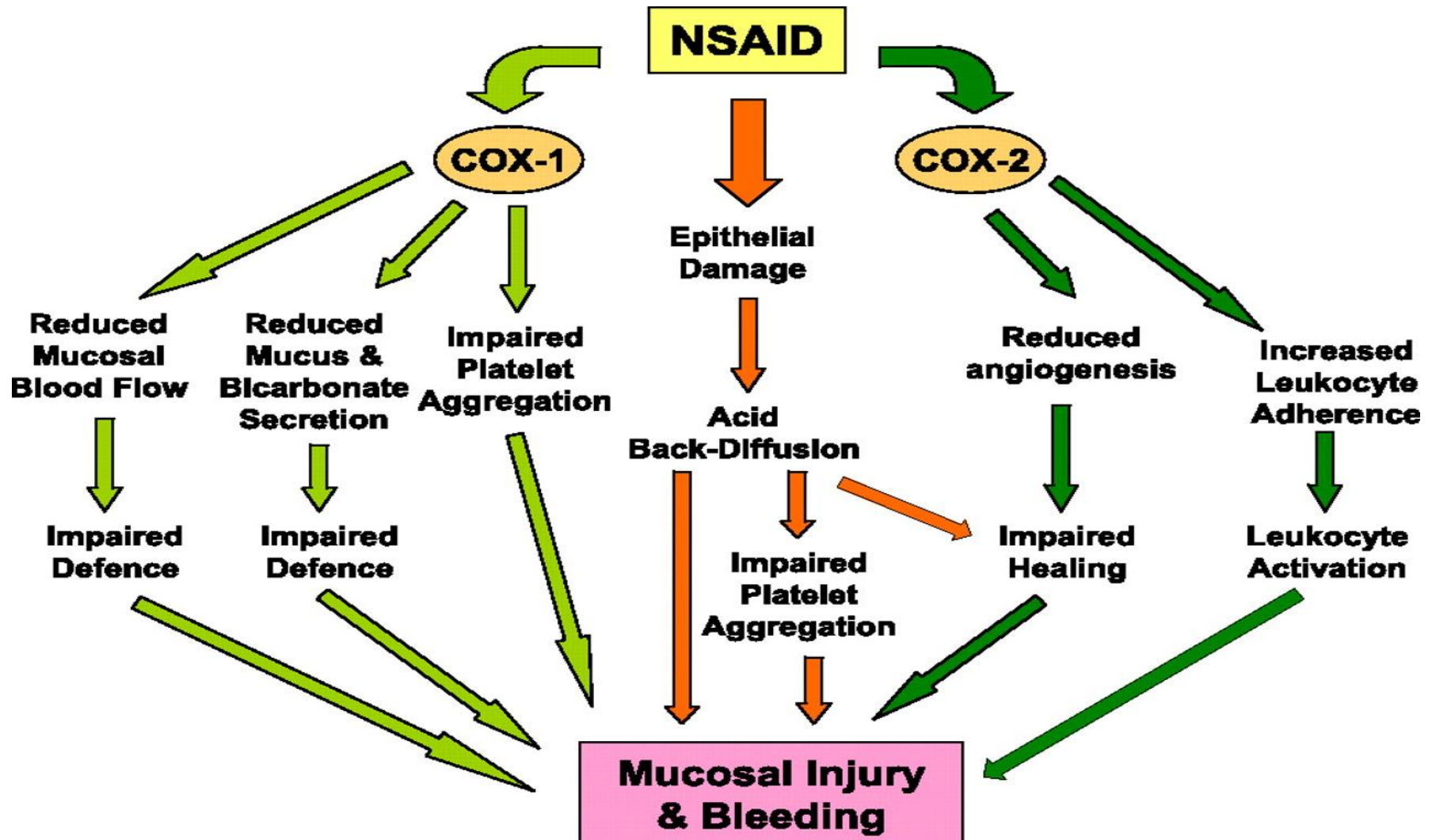
Mechanisms

NSAIDs 2

- Coexisting *H. pylori* infection increases the likelihood and intensity of NSAID-induced damage
- The annual risk of a life-threatening ulcer-related complication is (1 – 4)% in patients who use NSAIDs long-term, with older patients at the highest risk
- NSAID use is responsible for approximately one half of perforated ulcers.

Mechanisms

NSAIDs



Mechanisms

Zollinger- Ellison Syndrome 1

- The classic triad of Zollinger-Ellison syndrome involves peptic ulcers in unusual locations (i.e., the jejunum), massive gastric acid hypersecretion, and a gastrinproducing islet cell tumor of the pancreas (gastrinoma)
- Gastrinoma in the pancreas appears in approximately 50% of patients

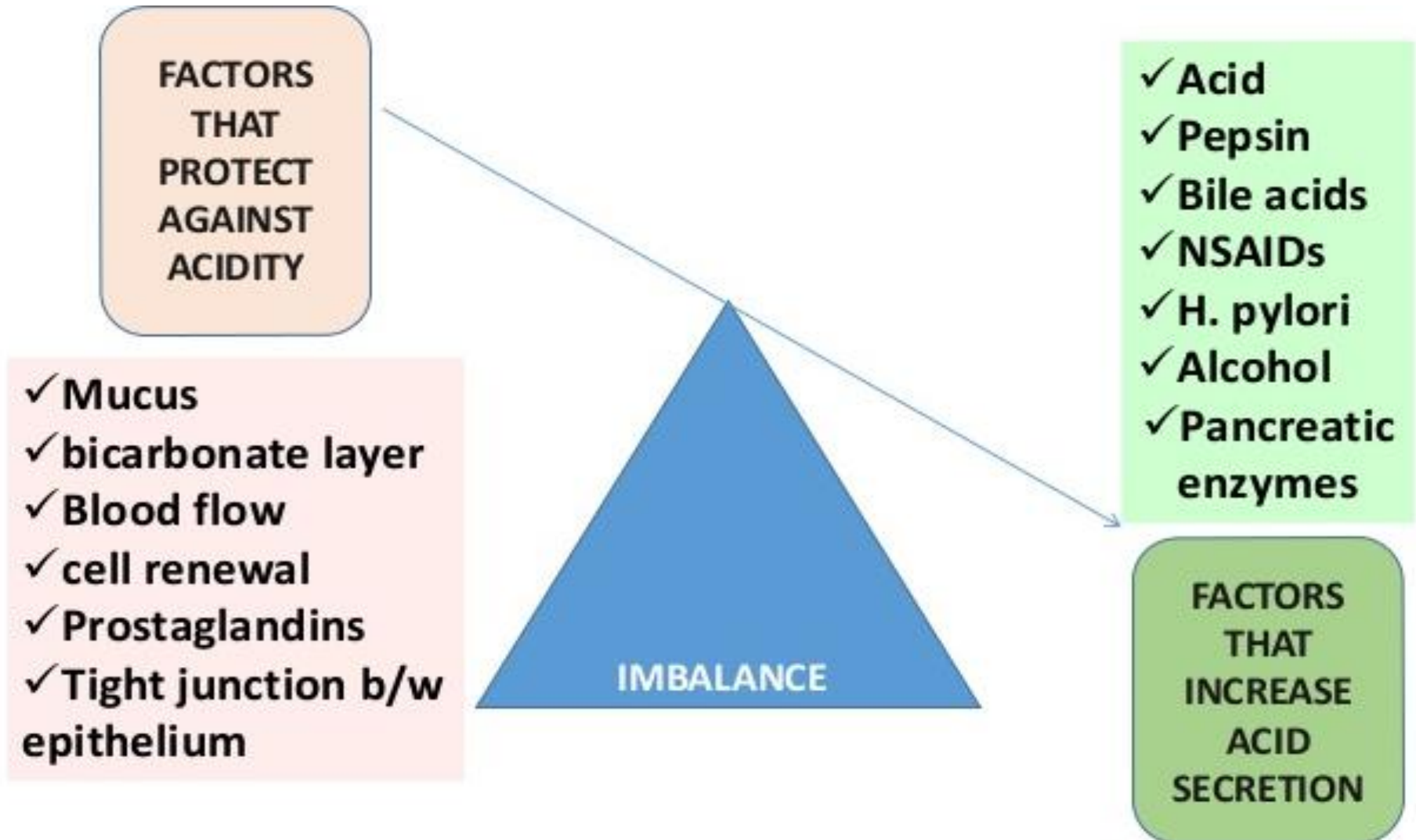
Mechanisms

Zollinger- Ellison Syndrome 2

- Another 20% of patients have it in the duodenum and others have it in the stomach, peripancreatic lymph nodes, liver, ovary, or small-bowel mesentery
- Zollinger-Ellison syndrome accounts for only 0.1% of all duodenal ulcer disease.

Mechanisms

Protective vs. Hostile Factors



US MLE TEST

A 34-year-old Nigerian born female presents with burning in her epigastric region. Ultimately, esophagoduodenoscopy (EGD) demonstrates an ulcer in the proximal duodenum. Which of the following treatments would best address the cause of her ulcer?

1. Antibiotics, 2. Cessation of NSAID use, 3. Surgical resection, 4. Cimetidine, 5. Omeprazole.

US MLE TEST EXPLANATION

The correct answer is 1. Based on the patient's presentation and the finding of a duodenal ulcer, the most likely cause is an H. pylori infection (most common cause of peptic ulcer disease). Therefore, antibiotics would be the best treatment.

Incorrect Answers:

2: Though NSAID use can mediate peptic ulcer disease, NSAID use is not the most common cause of peptic ulcer disease., 3: Surgical resection might prove useful in those suffering from Zollinger-Ellison syndrome, however Zollinger-Ellison syndrome is not the most common cause of peptic ulcer disease., 4 and 5: Though cimetidine (H₂ receptor blocker) and omeprazole (blocks H release) would be effective in lowering the acidity of the stomach, stomach acidity itself is not the most likely cause of peptic ulcer disease.

Classification

(International Classification of Diseases (ICD))

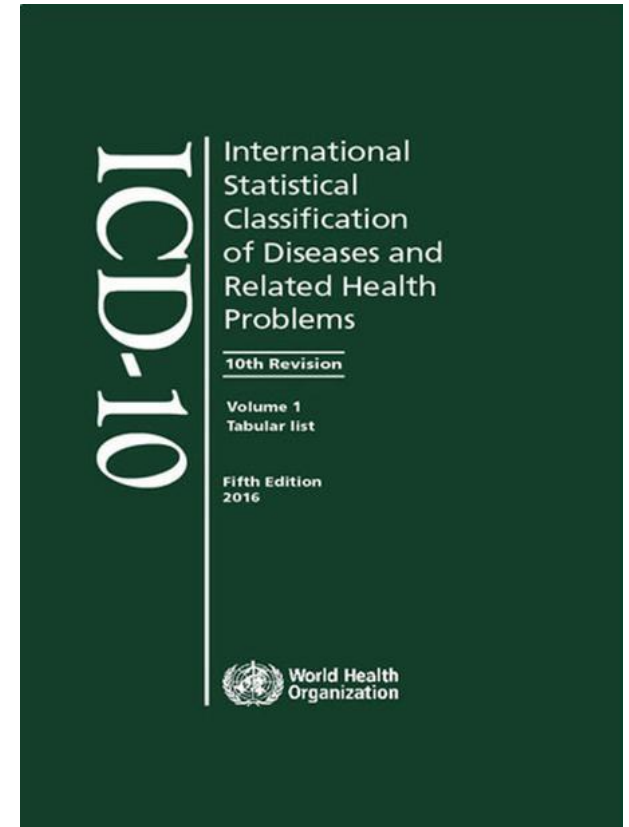
XI Diseases of the digestive system

K25 Gastric ulcer

K26 Duodenal ulcer

K27 Peptic ulcer, site unspecified

K28 Gastrojejunal ulcer



Classification

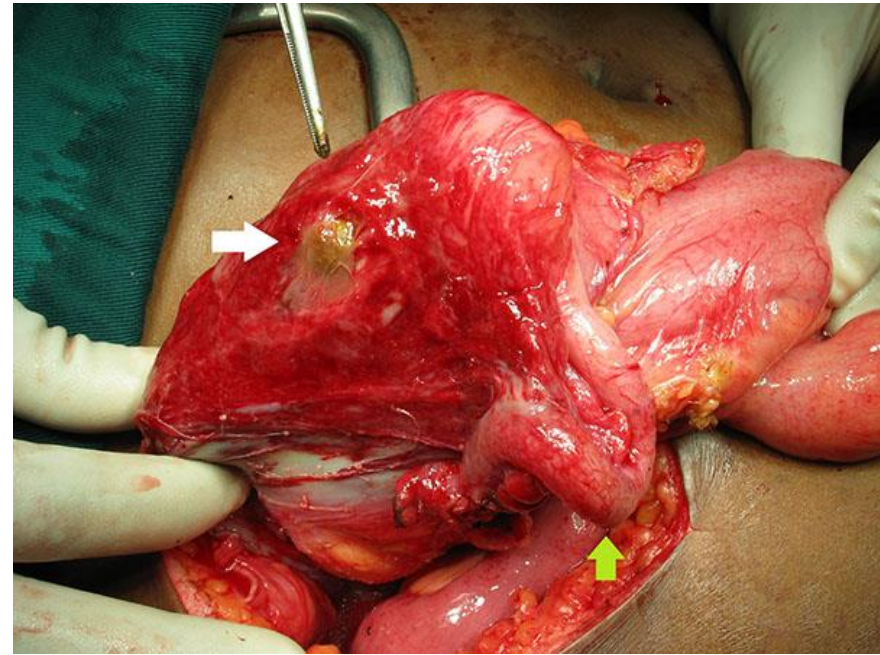
(Inflammatory) Stages

Stages	Manifestation
Active stage	
A1	The surrounding mucosa is edematously swollen and no regenerating epithelium is seen endoscopically
A2	The surrounding edema has decreased, the ulcer margin is clear, and a slight amount of regenerating epithelium is seen in the ulcer margin. A red halo in the <u>marginal</u> zone and a white slough circle in the ulcer margin are <u>frequently</u> seen. Usually, converging mucosal folds can be followed right up to the ulcer margin
Healing stage	
H1	The white coating is becoming thin and the regenerating epithelium is extending into the ulcer base. The gradient between the ulcer margin and the ulcer <u>floor</u> is becoming flat. The ulcer crater is still evident and the margin of the ulcer is sharp. The diameter of the mucosal defect is about one-half to two-thirds that of A1
H2	The defect is smaller than in H1 and the regenerating epithelium covers most of the ulcer floor. The area of white coating is about a quarter to one-third that of A1
Scarring stage	
S1	The regenerating epithelium <u>completely</u> covers the floor of ulcer. The white coating has disappeared. Initially, the regenerating region is markedly red. Upon close observation, many <u>capillaries</u> can be seen. This is called "red scar"
S2	In several months to a few years, the redness is reduced to the color of the surrounding mucosa. This is called "white scar"

Classification

Morphological by Area

- Duodenum (duodenal ulcer)
- Esophagus (esophageal ulcer)
- Stomach (gastric ulcer)
- Meckel's diverticulum (Meckel's diverticulum ulcer; is very tender with palpation).



Perforated diverticulum
with inflamed mass

Classification

Morphological by Johnson 1

Type I: Ulcer along the body of the stomach, most often along the lesser curve at incisura angularis along the locus minoris resistentiae, not associated with acid hypersecretion

Type II: Ulcer in the body in combination with duodenal ulcers, associated with acid oversecretion

Classification

Morphological by Johnson 2

Type III: In the pyloric channel within 3 cm of pylorus, associated with acid oversecretion

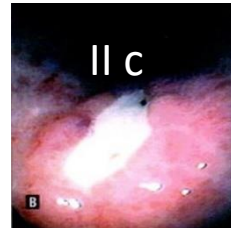
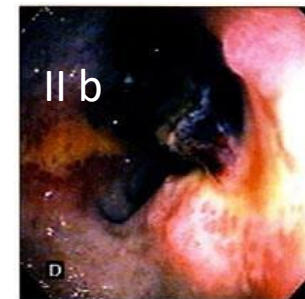
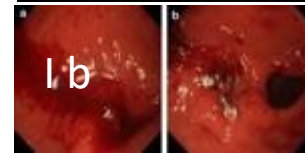
Type IV: Proximal gastroesophageal ulcer

Type V: Can occur throughout the stomach, associated with chronic use of NSAIDs.

Classification

Forest endoscopic classification 1

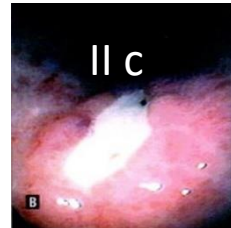
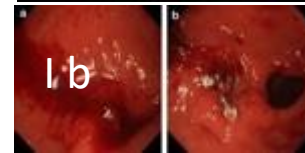
- Acute hemorrhage
 - Forrest I a (Spurting hemorrhage)
 - Forrest I b (Oozing hemorrhage)
- Signs of recent hemorrhage
 - Forrest II a (Visible vessel)
 - Forrest II b (Adherent clot)
 - Forrest II c (Hematin on ulcer base))



Classification

Forest endoscopic classification 2

- Lesions without active bleeding
 - Forrest III (Clean base)



Signs and Symptoms 1

- Abdominal pain, classically epigastric correlated to mealtimes
- In case of duodenal ulcers the pain appears about 3 hours after taking a meal
- Bloating and abdominal fullness
- Water brash
- Nausea, and copious vomiting
- Loss of appetite and weight loss

Signs and Symptoms 2

- Hematemesis (vomiting of blood)
- Melena (tarry, foul-smelling feces due to presence of oxidized iron from hemoglobin)
- Rarely gastric or duodenal perforation, which leads to acute peritonitis, extreme, stabbing pain, and requires immediate surgery
- Pyloric outlet obstruction.

Physical Examination 1

- Uncomplicated PUD: epigastric and right upper quadrant tenderness, guaiac-positive stool resulting from occult blood loss, melena resulting from acute or subacute gastrointestinal bleeding, succussion splash resulting from partial or complete gastric outlet obstruction

Physical Examination 2

- Patients with perforated PUD usually present with a sudden onset of severe, sharp abdominal pain; as even slight movement can tremendously worsen their pain, these patients assume a fetal position; abdominal examination discloses generalized tenderness, rebound tenderness, guarding, and rigidity

Physical Examination 3

- Patients with perforated PUD may also demonstrate signs and symptoms of septic shock, such as tachycardia, hypotension, and anuria; these indicators of shock may be absent in elderly or immunocompromised patients or in those with diabetes.

Red Flags

- Bleeding or anemia
- Early satiety
- Unexplained weight loss
- Progressive dysphagia or odynophagia
- Recurrent vomiting
- Family history of gastrointestinal cancer



Hemorrhage due to
Duodenal Ulcer

USMLE TEST

A 45-year-old male patient with a history of recurrent nephrolithiasis and chronic lower back pain presents to the ER with severe, sudden-onset, upper abdominal pain. The patient is febrile, hypotensive, and tachycardic, and is rushed to the OR for exploratory laparotomy. Surgery reveals that the patient has a perforated gastric ulcer. Despite appropriate therapy, the patient expires, and subsequent autopsy reveals multiple ulcers in the stomach, duodenum, and jejunum. The patient had been complaining of abdominal pain and diarrhea for several months but had only been taking ibuprofen for his lower back pain for the past 3 weeks. What is the most likely cause of the patient's presentation?

1. A gastrin-secreting tumor of the pancreas,
2. A vasoactive-intestinal-peptide (VIP) secreting tumor of the pancreas,
3. Cytomegalovirus infection,
4. *H. pylori* infection,
5. Chronic NSAID use.

US MLE TEST EXPLANATION

The correct answer is 1. This patient with multiple, severe ulcers from his stomach to his small bowel most likely had Zollinger-Ellison syndrome (ZES), which is caused by a gastrin-secreting tumor.

Incorrect Answers:

2: VIP-secreting tumors may occur in MEN1, and often present with nausea, diarrhea, and abdominal pain. However, VIP suppresses gastric acid production, and peptic ulcers are not typically present., 3: Cytomegalovirus is associated with peptic ulcer formation, but does not typically cause multiple or jejunal ulcers., 4: H. pylori is one of the leading causes of peptic ulcers, but does not typically cause ulcers reaching into the jejunum., 5: Chronic NSAID use typically causes gastritis. Though it can cause peptic ulcer disease, it would not explain the severity of this patient's disease.

Complications 1

- Refractory, symptomatic peptic ulcer
- Internal bleeding
- Hemorrhage
- Anemia
- Obstruction (persist or recur despite endoscopic balloon dilation)
- Penetration
- Perforation

Complications 2

- Peritonitis
- Gastric malignancy

Endoscopy is the treatment of choice for bleeding ulcers.

Transcatheter arterial embolization is the treatment of choice for duodenal ulcers re-bleeding after therapeutic endoscopy or surgery.

Diagnosis 1

- The diagnosis is mainly established based on the characteristic symptoms
- Confirmation of the diagnosis is made with the help of tests such as endoscopies or barium contrast x-rays; the tests are typically ordered if the symptoms do not resolve after a few weeks of treatment, or when they first appear in a person who is over age 45 or who has other symptoms such as weight loss, because stomach cancer can cause similar symptoms

Diagnosis 2

- The diagnosis of *Helicobacter pylori* can be made by urea breath test; direct culture from an esophagogastroduodenoscopy (EGD) biopsy specimen; direct detection of urease activity in a biopsy specimen (rapid urease test); measurement of antibody levels in blood; stool antigen test; histological examination and staining of an EGD biopsy.

Diagnosis

Upper Gastrointestinal Endoscopy 1

- It is the preferred diagnostic test in the evaluation of patients with suspected gastric and duodenal ulcers
- Gastric ulcers appear as discrete mucosal lesions with a punched-out smooth ulcer base, which often is filled with whitish fibrinoid exudate

Diagnosis

Upper Gastrointestinal Endoscopy 2

- Most gastric ulcers tend to occur at the junction of the fundus and antrum, along the lesser curvature; benign ulcers tend to have a smooth, regular, rounded edge with a flat smooth base and surrounding mucosa that shows radiating folds; malignant ulcers usually have irregular heaped-up or overhanging margins; the ulcerated mass often protrudes into the lumen, and the folds surrounding the ulcer crater are often nodular and irregular

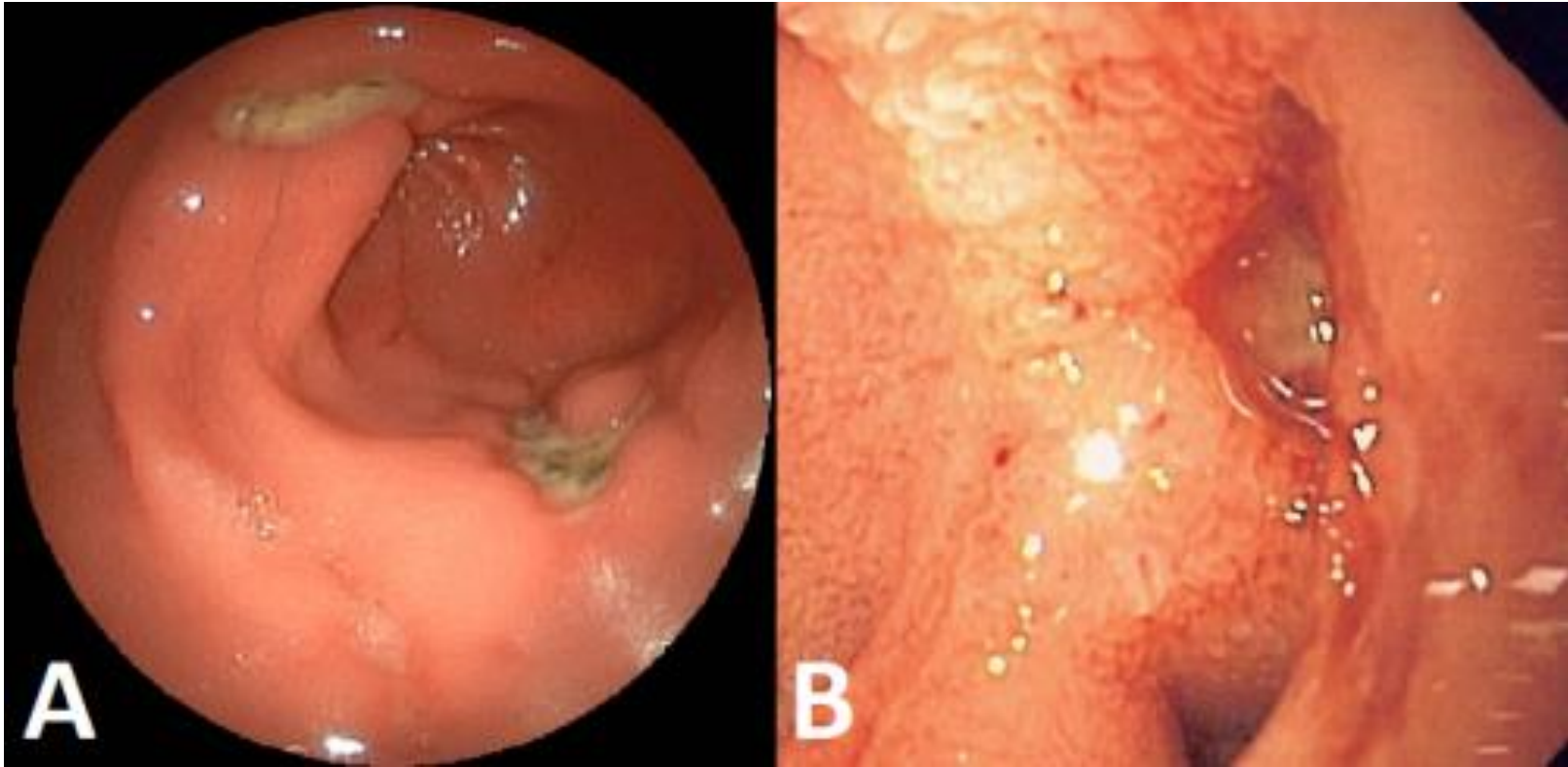
Diagnosis

Upper Gastrointestinal Endoscopy 3

- More than 95% of duodenal ulcers are found in the first part of the duodenum; most are less than 1 cm in diameter.

Diagnosis

Upper Gastrointestinal Endoscopy



Features of peptic ulcers on endoscopy (A) peptic ulcer located in the gastric antrum (B) hemorrhaging gastric ulcer.

Diagnosis

Upper Gastrointestinal Endoscopy



An ulcer (at upper center) in the wall of the duodenum, the first part of the small intestine. This ulcer is an open sore.

Diagnosis

Upper Gastrointestinal Endoscopy



A



B

Duodenal ulcers. A. Ulcer with a clean base. B. Ulcer with a visible vessel (arrow) in a patient with recent hemorrhage.

Diagnosis

Barium X-ray or Upper Gastrointestinal series 1

- Barium x-ray or upper GI series is a widely available and accepted method to establish a diagnosis of peptic ulcer in the stomach or duodenum
- Though less invasive than endoscopy, the barium x-ray is limited by being less sensitive and accurate at defining mucosal disease, or distinguishing benign from malignant ulcer disease

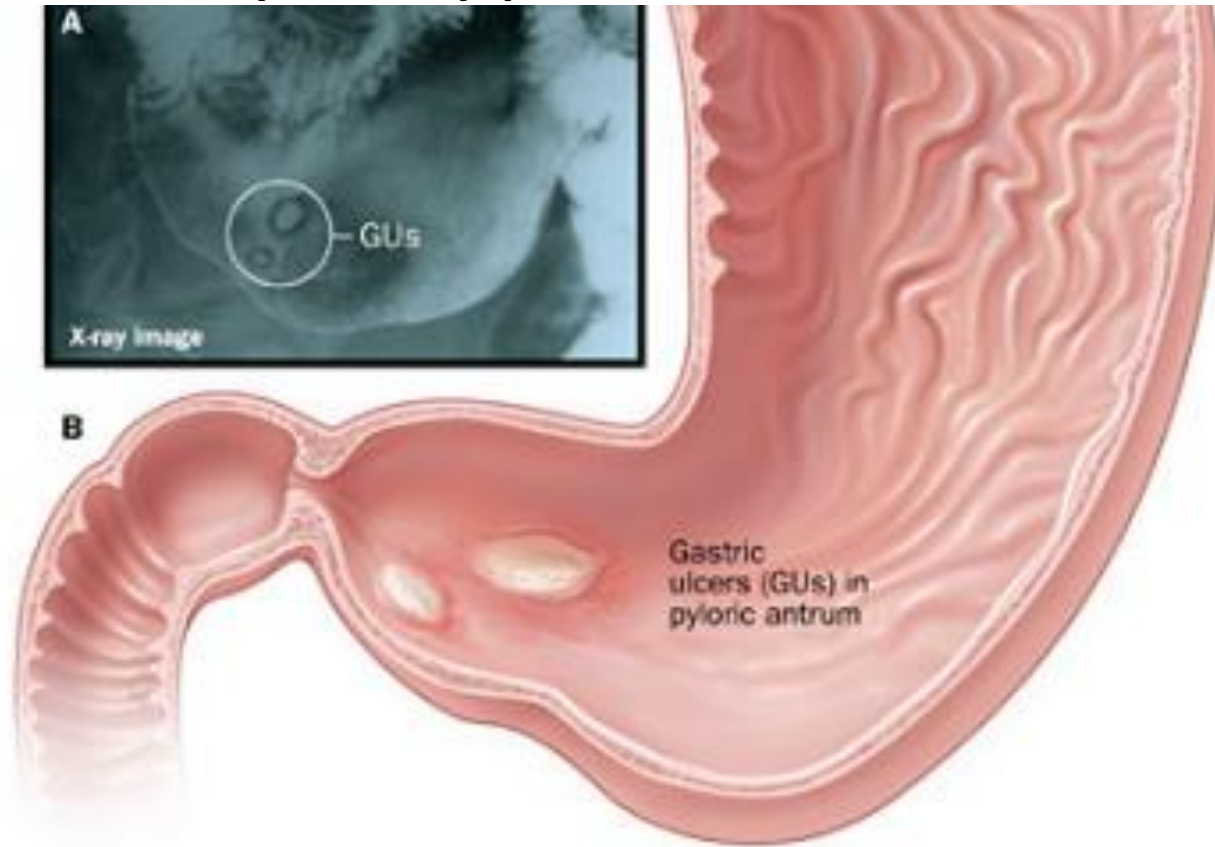
Diagnosis

Barium X-ray or Upper Gastrointestinal series 2

- In patients who have anatomic deformities from previous gastric surgery or scarring from chronic inflammation, barium x-rays may be difficult to interpret
- Generally, these x-rays have up to a 30% false negative and a 10% false positive rate.

Diagnosis

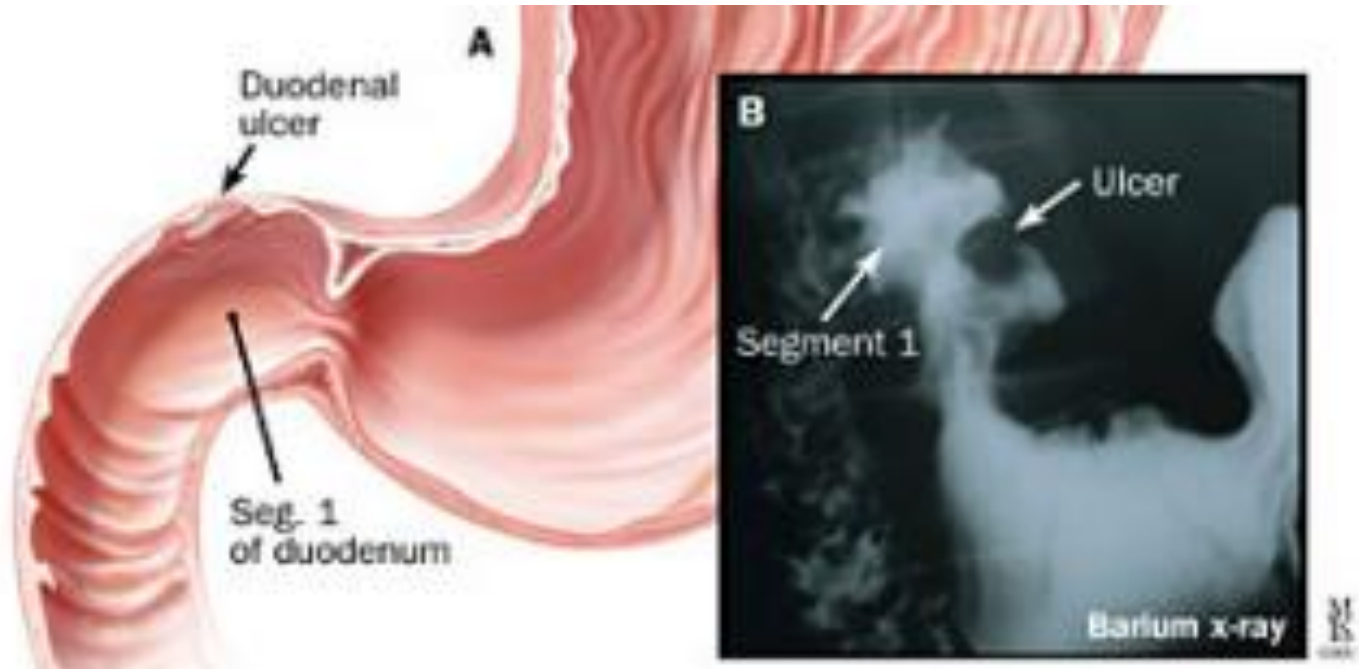
Barium X-ray or Upper Gastrointestinal series



X-ray of gastric ulcer in the antrum; B, corresponding illustration of a gastric ulcer.

Diagnosis

Barium X-ray or Upper Gastrointestinal series

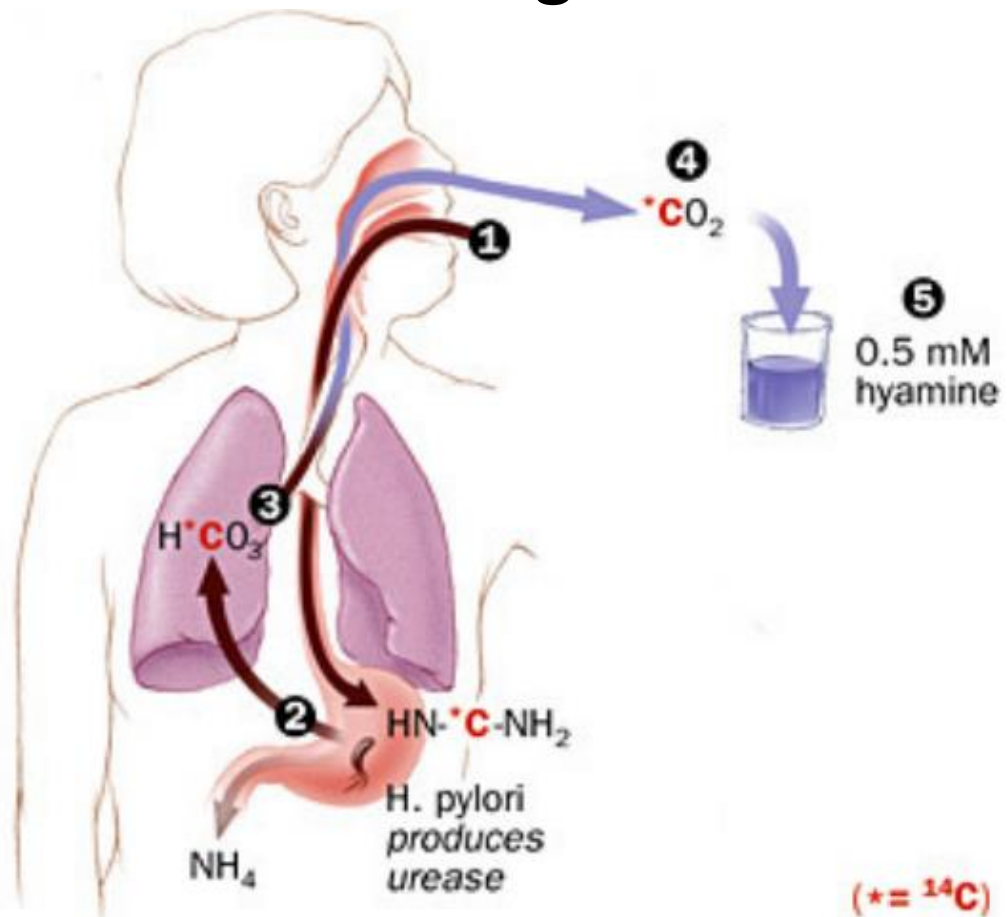


Duodenal ulcer; B, corresponding x-ray.

Diagnosis

Urea Breath Testing

1. Patient drinks $\text{HN}^*\text{C}-\text{NH}_2$.
In the stomach, $\text{HN}^*\text{C}-\text{NH}_2$ is broken down by urease into H^*CO_3 and NH_4 .
2. H^*CO_3 travels to the lung and is...
3. ...expired...
4. ... as $^*\text{CO}_2$ into...
5. ... a 0.5 mM hyamine solution, where a scintillation cocktail is added to test for $^*\text{C}$.



Urea breath test determines the presence of *H. pylori*.

Differential Diagnosis 1

- Acute cholangitis
- Acute coronary syndrome
- Acute and chronic gastritis
- Cholecystitis and biliary colic (in emergency medicine)
- Diverticulitis
- Emergent treatment of gastroenteritis
- Esophageal rupture and tears in emergency medicine

Differential Diagnosis 2

- Esophagitis
- Gallstones (cholelithiasis)
- Gastroesophageal reflux disease
- Inflammatory bowel disease
- Viral hepatitis

US MLE TEST

A 40-year-old man presents to clinic three weeks after undergoing a total hip replacement. He complains of chronic nausea, epigastric pain and occasional melena, and notes that he has been taking celecoxib for pain control since his surgery. An esophagogastroduodenoscopy is performed, and a biopsy is taken of an erythematous area of the antrum of the stomach (Figure A). What treatment is recommended in this patient?

1. Sulfasalazine and corticosteroid therapy,
2. Gluten free diet
3. Omeprazole, clarithromycin and amoxicillin,
4. Intramuscular intrinsic factor injection,
5. Discontinue celecoxib.

US MLE TEST EXPLANATION

The correct answer is 3. The clinical presentation is consistent with type B chronic gastritis, most commonly due to *H. pylori* infection, which should be treated with a combination of acid suppression and antibiotics.

Incorrect Answers:

1: Sulfasalazine and corticosteroid therapy would be indicated in patients with inflammatory bowel disease., 2: Gluten free diet would be recommended for patients with Celiac disease., 4: Intramuscular intrinsic factor injection would be indicated in Type A chronic gastritis, which is characterized by the presence of autoantibodies directed against the patient's parietal cells. The lack of intrinsic factor can lead to megaloblastic anemia if not corrected., 5: NSAIDs that target cyclooxygenase-1 are associated with chronic gastritis. COX-2 inhibitors (celecoxib) have been shown to have less gastrointestinal side effects than COX-1 inhibitors and *H. pylori* infection is more likely a cause for this patient's gastritis given the presence Gram negative rods on biopsy.

Management

Approach Considerations 1

- Treatment PUD varies depending on the etiology and clinical presentation
- Treatment options include antisecretory therapy, triple therapy for *H pylori* infection, endoscopy followed by appropriate therapy based on findings, and *H pylori* serology followed by triple therapy
- Endoscopy performed 6-8 weeks after the diagnosis of PUD is required to document healing of ulcers and to rule out gastric cancer

Management

Approach Considerations 2

- Documentation of *H pylori* cure with a noninvasive test, such as the urea breath test or fecal antigen test, is appropriate in patients with complicated ulcers.
- Perform endoscopy early in patients older than 45-50 years and in patients with associated so-called alarm symptoms, such as dysphagia, recurrent vomiting, weight loss, or bleeding.

Management

Lifestyle Changes 1

- In the past, it was common practice to tell people with PUD to consume small, frequent amounts of bland foods
- Exhaustive research has shown that a bland diet is not effective in reducing the incidence or recurrence of ulcers
- Large amounts of food should still be avoided, because stretching the stomach can result in painful symptoms

Management

Lifestyle Changes 2

- A diet rich in fiber may speed the healing of existing ulcers
- Probiotics, which are "good" bacteria added to yogurt and other fermented milk drinks, may protect the gastrointestinal system
- Some evidence suggests that exercise may help reduce the risk for ulcers in some people
- Stress relief programs have not been shown to promote ulcer healing, but they may have other health benefits.

Management

Patients not taking NSAIDs 1

- If an ulcer is seen (endoscopy) and the patient is infected with *H. pylori*, treatment for the infection is started, followed by 4 to 8 weeks of treatment with a proton pump inhibitor
- If an ulcer is seen but *H. pylori* is not present, patients are usually treated with PPI for 8 weeks
- If no ulcer is seen and the patient is not infected with *H. pylori*, the first treatment attempt will usually be with PPI

Management

Patients not taking NSAIDs 2

- If the PPI dose is not effective, occasionally doubling the dose will relieve symptoms
- Those who do not respond to treatment, or whose symptoms return relatively quickly, will often need an upper endoscopy
- Using antibiotics when there is no clear evidence of ulcers will lead to unnecessary antibiotic prescriptions and increase the risk for side effects.

Management

The Treatment of *H. Pylori* 1

- Reported cure rates for *H. pylori* range from 70 - 90% after antibiotic treatment
- The standard treatment regimen uses two antibiotics (clarithromycin, amoxicillin) and a PPI (omeprazole, lansoprazole, esomeprazole, and rabeprazole)
- PPIs are important for all types of peptic ulcers, and are a critical partner in antibiotic regimens; they reduce acidity in the intestinal tract, and increase the ability of antibiotics to destroy *H. pylori*

Management

The Treatment of *H. Pylori* 2

- Some doctors substitute the antibiotic metronidazole for either clarithromycin or amoxicillin
- Patients typically take this combination treatment for at least 14 days
- Many studies, however, suggest that a 7-day treatment may work just as well.

Management

NSAID-Induced Ulcers 1

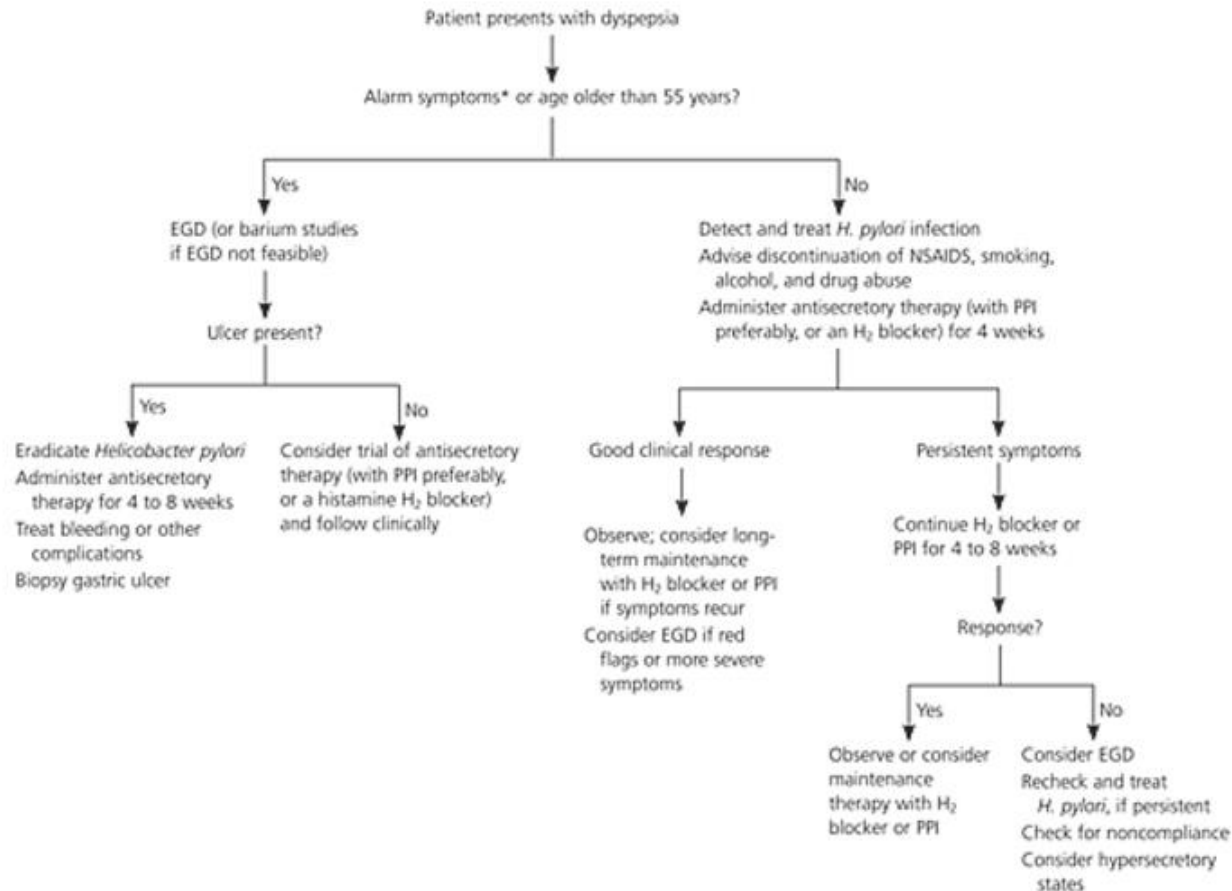
- Patients who are beginning long-term NSAID therapy should first be tested for *H pylori*
- NSAIDs should be immediately discontinued in patients with positive *H pylori* test results if clinically feasible
- For patients who must continue with their NSAIDs, PPI maintenance is recommended to prevent recurrences even after eradication of *H pylori*

Management

NSAID-Induced Ulcers 2

- Active ulcers associated with NSAID use are treated with an appropriate course of PPI therapy and the cessation of NSAIDs
- For patients with a known history of ulcer and in whom NSAID use is unavoidable, the lowest possible dose and duration of the NSAID and co-therapy with a PPI or misoprostol are recommended.

Management Schematic Diagram



Algorithm for the treatment of peptic ulcer disease (PPI - proton pump inhibitor).

Management

Refractory Ulcers 1

- Refractory PUD (i.e., disease that fails to heal after eight to 12 weeks of therapy) may be caused by persistent or resistant *H. pylori* infection, continued NSAID use, giant ulcers requiring longer healing time, cancer, tolerance of or resistance to medications, or hypersecretory states
- Therapy for refractory PUD involves treatment of the underlying cause and prolonged administration of standard doses of a proton pump inhibitor

Management

Refractory Ulcers 2

- Up to 25% of patients with gastric ulcers who continue to take NSAIDs may require PPI therapy for longer than eight weeks.

Management

Surgery 1

- Surgery is indicated in patients who are intolerant of medications or do not comply with medication regimes, and those at high risk of complications (e.g., patients dependent on steroids or NSAIDs, those with giant ulcer, those with ulcers that fail to heal with treatment)
- Surgery should also be considered for patients who have a relapse during maintenance treatment or who have had multiple courses of medications

Management

Surgery 2

- Surgical options for duodenal ulcers include truncal vagotomy and drainage (pyloroplasty or gastrojejunostomy), selective vagotomy (preserving the hepatic and celiac branches of the vagus) and drainage, highly selective vagotomy, or partial gastrectomy
- The indications for urgent surgery include failure to achieve hemostasis endoscopically, recurrent bleeding despite endoscopic attempts at achieving hemostasis, and perforation.

US MLE TEST

A 41-year-old female complains of frequent diarrhea and abdominal pain between meals. Endoscopy reveals a duodenal ulcer distal to the duodenal bulb. CT scan of the abdomen demonstrates a pancreatic mass, and subsequent tissue biopsy of the pancreas reveals a malignant islet cell tumor. Which of the following hormones is likely to be markedly elevated in this patient?

1. Gastrin, 2. Cholecystokinin, 3. Secretin, 4. Vasoactive intestinal peptide, 5. Motilin.

US MLE TEST EXPLANATION

The correct answer is 1. The patient is suffering from Zollinger-Ellison (ZE) syndrome due to a pancreatic gastrinoma. Excess gastrin secretion leads to excess gastric acid production in the stomach, often resulting in peptic ulcer disease.

Incorrect Answers:

2: Cholecystokinin regulates gallbladder contraction, gastric emptying, and pancreatic secretion. It is secreted by I cells in the duodenum and jejunum in response to fatty acids and amino acids., 3: Secretin is secreted by duodenal S cells. It decreases gastric acid secretion and increases pancreatic bicarbonate secretion., 4: Vasoactive intestinal peptide increases intestinal water and electrolyte secretion and prompts relaxation of intestinal smooth muscle., 5: Motilin is secreted by the small intestine and is an important regulator of peristalsis.

Prognosis 1

- Typically, duodenal ulcers heal in 4 weeks and gastric ulcers in 8 weeks with PPI therapy
- For patients with peptic ulcers caused by *H pylori*, the prognosis after *H pylori* eradication is good: less than 20% will experience recurrence, and this is lower for duodenal ulcers than for gastric ulcers
- *H pylori* eradication is also beneficial in those with complicated ulcer disease

Prognosis 2

- For ulcers associated with NSAID use, discontinuing the NSAID (and eradicating *H pylori* if present) will lead to a low rate of ulcer recurrence
- In patients who continue using NSAIDs, ulcer recurrence is high, and thus co-prescription of a PPI is advisable.

Prophylaxis

- There is no proven way to prevent peptic ulcer disease. But several lifestyle changes may reduce the risk of ulcers
- Curb NSAID use
- Get tested for *H. pylori*
- Stop smoking
- Reduce distress
- Watch alcohol intake.

Abbreviations

CBC - complete blood count

H. pylori - *Helicobacter pylori*

EGD – esophagogastroduodenoscopy

NSAIDs - non-steroidal anti-inflammatory drugs

PPI - proton pump inhibitor

ZES - Zollinger-Ellison syndrome

Diagnostic and treatment guidelines

- [Evidence-based clinical practice guidelines for peptic ulcer disease 2015](#)
- [Peptic Ulcer Disease Treatment & Management](#)
- [Peptic Ulcer Disease](#)
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